به نام خداوند جان آفرین

مراقبت های ویژه در بیماری های زنان و مامایی

نسیم بهرامی
عضو هیات علمی دانشکده پرستاری و مامایی

Every Mom's Dream.....

دکتر نسیم بهرامی

The 99 obstetric admissions to ICU were represented by 0.2% of all deliveries and 1.6% of all ICU admissions.

Most patients were young (median age of 30 years) with parity (median) of 3 and stayed in ICU for an average of 2 days. All except one patient were admitted during the postpartum period. The majority (76%) were admitted after cesarean section.

Obstetric hemorrhage (32%) and hypertension (29%) were the 2 most common indications for admission. The majority (59/99) of patients also had pre-existing medical problem and most common (16/59) was rheumatic heart disease.

Preeclampsia (23/99) and eclampsia 10/99 were the most frequent obstetric complication. Thirty-six percent of our patients required ventilatory support. In the majority of patients, direct arterial (81%) and central venous (73%) pressure monitoring was carried out. Pulmonary arterial and left atrial pressure was monitored in 4%.

Almost one third of patients received antihypertensive therapy. Inotropic support was given to 9% and blood (and its products) was given to 46% of patients. Antibiotics (28%) and magnesium sulphate (25%) were the most frequently used medicines.

Out of the total 99 admissions, one patient died and 16 patients developed complications.

- A total of 43 women required admission to the intensive care unit (ICU), which represented 0.37% of all deliveries.
- The majority (95.3%) of patients were admitted to the ICU postpartum. The most common reasons for admissions were (pre) eclampsia (48.8%) and obstetric haemorrhage (37.2). The remainder included adult respiratory distress syndrome (6.9%), pulmonary embolism (2.3%) and neurological disorders (4.6%).
- Mechanical ventilation was required to support 18.6% of patients and transfusion of red blood cells was needed for 48.8% of patients.
- There were three maternal deaths (6.9%).
- A multidisciplinary team approach is essential to improve the management of hypertensive disorders and postpartum haemorrhage to achieve significant improvements in maternal outcome.

Introduction…

- The commonest indication for Intensive Care Unit (ICU) admission of obstetric patients is hemorrhage, both ante-partum and post-partum.
- Hypertensive disorders, pre-eclampsia, and its related complications are also major contributory factors for such admissions.
- These reflect the lack of proper antenatal care and timely management of obstetrical emergencies, especially in the developing countries
- The obstetrician's involvement is of prime importance when managing such cases in the ICU

Challenges…

- Economic factors, socio-cultural characteristics, and different hospital protocols and management policies, further widen the gap of bringing uniform admission criteria
- One of the most striking similarities in all such patients is their young age, which in fact is a good prognostic indicator, provided they receive timely interventions for their acute pathologies

Indications for ICU admission

- Conditions related to pregnancy – eclampsia, severe pre-eclampsia, hemorrhage, amniotic fluid embolism, acute fatty liver, peri-partum cardiomyopathy, aspiration syndromes, infections etc.

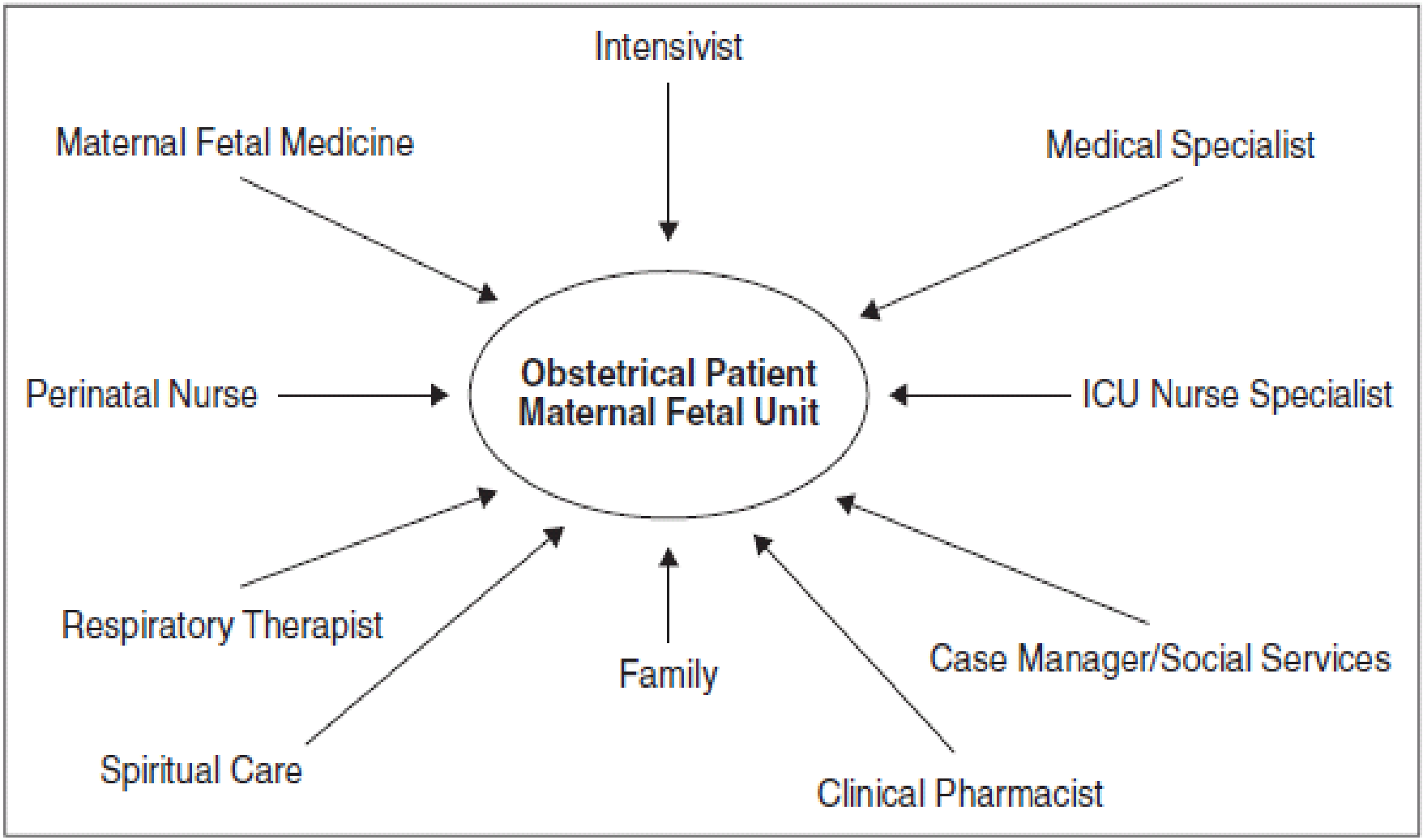
- Medical diseases that may be aggravated during pregnancy - congenital heart diseases, rheumatic and non-rheumatic valvular diseases, pulmonary hypertension, anemia, renal failure etc.

- Conditions that are not related to pregnancy – trauma, asthma, diabetes, autoimmune diseases etc.

MONITORING DURING CRITICAL ILLNESS

- Invasive monitoring is essential in most critically ill patients both during surgical procedures and during the ICU stay.
- Central venous pressure monitoring is used to guide fluid administration
- The pulmonary artery catheter is extremely useful for measuring central venous pressure (CVP), pulmonary capillary wedge pressure (PCWP), systemic vascular resistance (SVR), cardiac output (CO), pulmonary artery (PA) pressure, and mixed venous oxygen saturation (SvO_2).

Staff pattern to help the skilled and experienced obstetrician IN ICU



Relevance of OB physiology

- 5-10 % of women are pregnant
  - Many don't know or show
- Any female of reproductive age could be pregnant
  - Should be assumed so!
- Virtually every organ system affected
- Can touch almost any specialty

CASE HISTORY

Case 1

- 36 y.o. female presents to ER
- CC: Fatigue, dyspnea, chest pain
- HPI:
  - Progressive dyspnea over several weeks.
  - Poor exercise tolerance and easy fatigability
    - 'get winded after 1 flight of stairs'
  - Substernal chest pain, peaks in morning and night
  - Nocturnal cough, semi-productive – clear
  - Leg swelling
  - polyuria

Case 1

- PMH
  - Mild obesity
- Ob/gyn – menses at age 12; irregular menses; no pregnancies
- Meds
  - Oral contraceptives
  - multivitamins
- Social
  - Married for 2 years. No exposures

Case 1: PE

- Skin
  - warm, clammy. Mild facial acne and increased hair – medium coarseness
- HEENT
  - Nasal mucosa slightly hyperemic.
  - Mild non-nodular thyromegaly
- CV
  - Tachycardia (HR 107)
  - + JVD
  - systolic murmurs over pulmonic and aortic v.

PE cont'd

- Chest
  - Clear bilaterally. Diaphragm elevated
  - 1+ pretibial pitting edema

- Abd
  - Skin – spider angiomata and striae. Medium course hair, infraumbilical.
  - Distended, firm, non-tender.

Studies / labs

- EKG:
  - Sinus rhythm; tachy; Left axis deviation
- CXR:
  - Lungs clear. Cardiomegaly.
  - Labs:
  - Hct 32% (low); WBC 12 (high)
  - Cholesterol 300 mg/dl
  - D-dimer elevated
  - Potassium and creatinine low

WHAT DOES SHE HAVE???

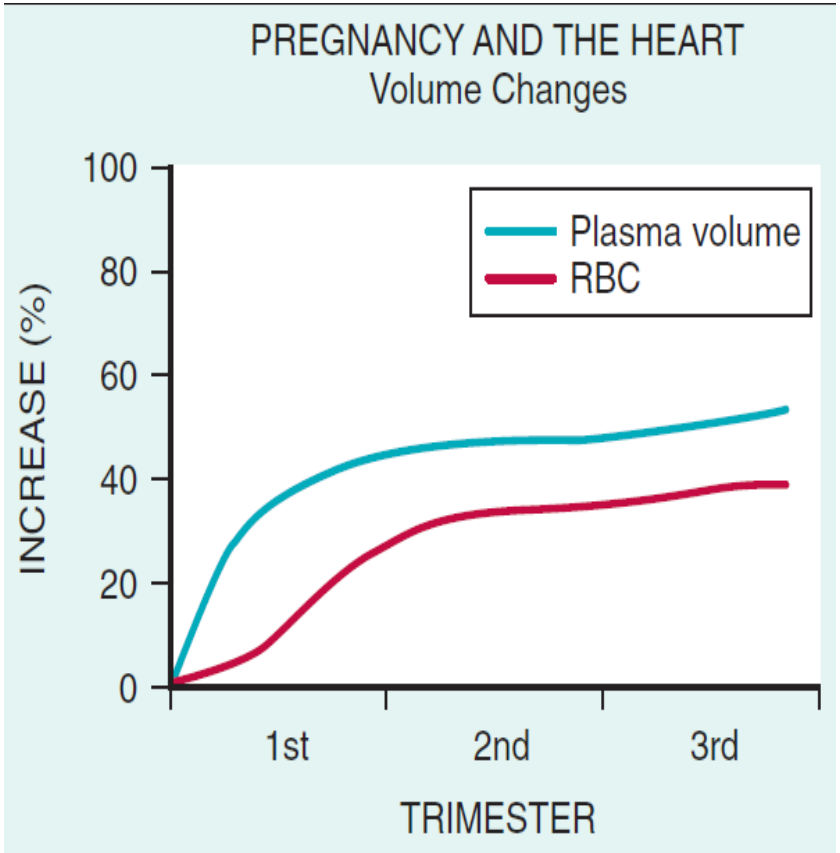
General Principles

- Most changes begin early
  - Even before pregnancy recognized
- Most are hormonally driven
  - Progesterone, estrogen, renin / aldosterone, cortisol, insulin
  - Some 'mechanically' driven
- Designed to optimize conditions for fetus & prepare for delivery
  - Delivery of oxygen & nutrients

PHYSIOLOGICAL CHANGES IN PREGNANCY

- Plasma Volume
- Heart Rate & Stroke Volume
- Cardiac Output
- BP
- Clinical Findings
- ECG

PLASMA VOLUME



- **Pl. volume start ↑ by 6 wks**
- **50% ↑ 2nd trimester then plateaus till delivery**
- **Red cell mass ↑ to lesser extent**

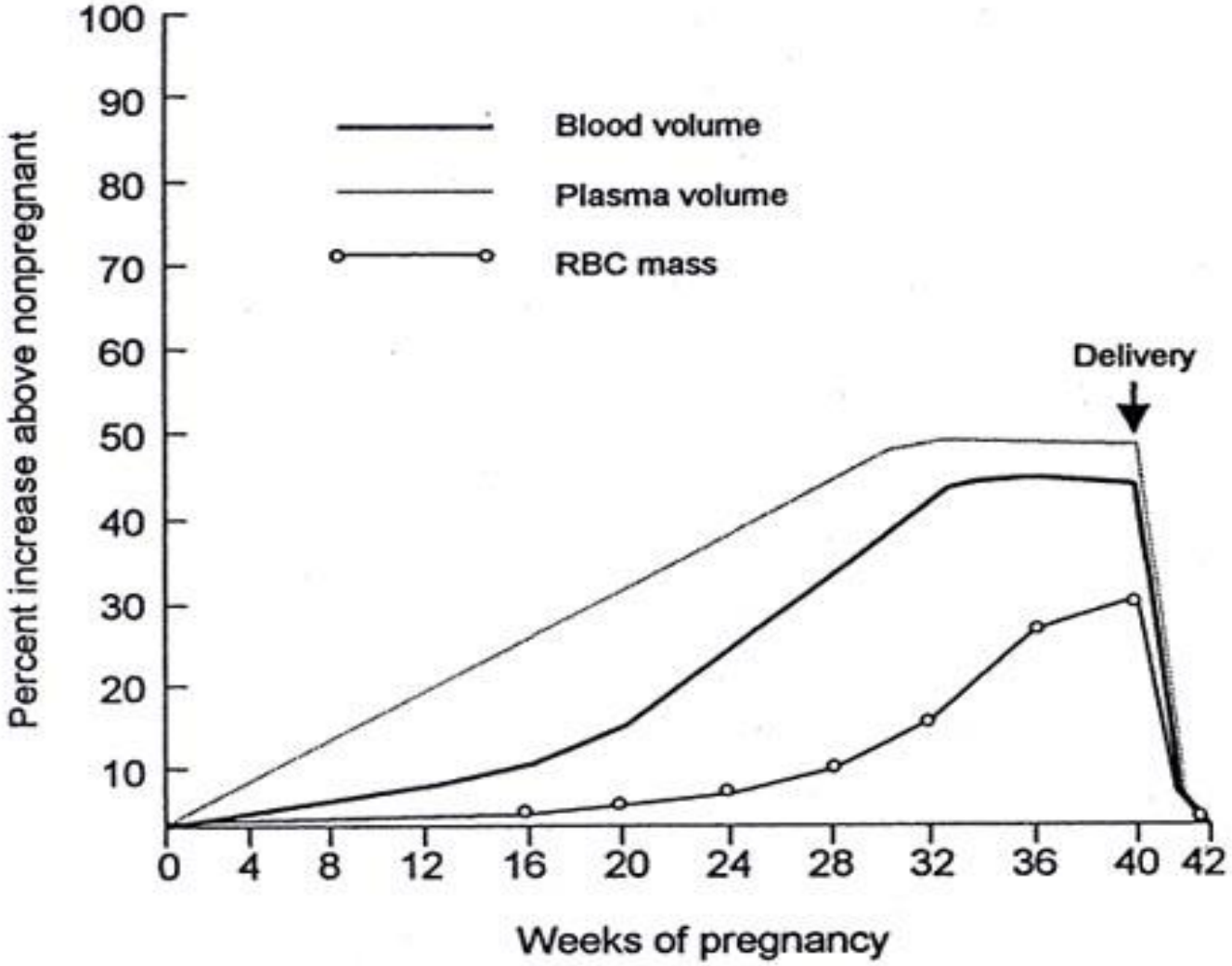
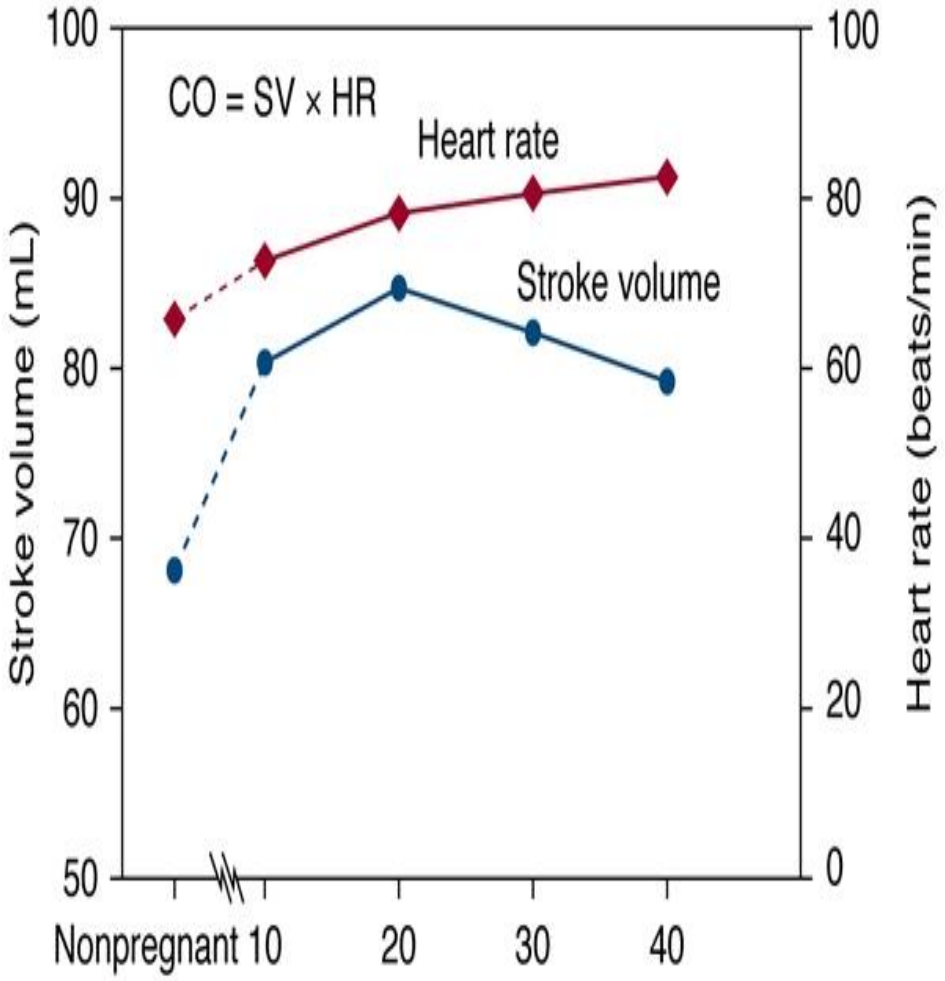


Figure 3–11. Blood volume changes during pregnancy. (From Scott D: Anemia during pregnancy. Obstet Gynecol Ann 1:219, 1972.)

Cardiovascular & Hematologic

- Vascular
  - Decreased tone / vaso-relaxation
    - SVR decreased **20%**
  - Positional effects
  - Placenta – low resistance shunt
- Hematologic
  - Blood volume increases 50-100%
  - RBC increases 25-40%
    - Relative anemia ("physiologic")

Heart rate & Stroke Volume

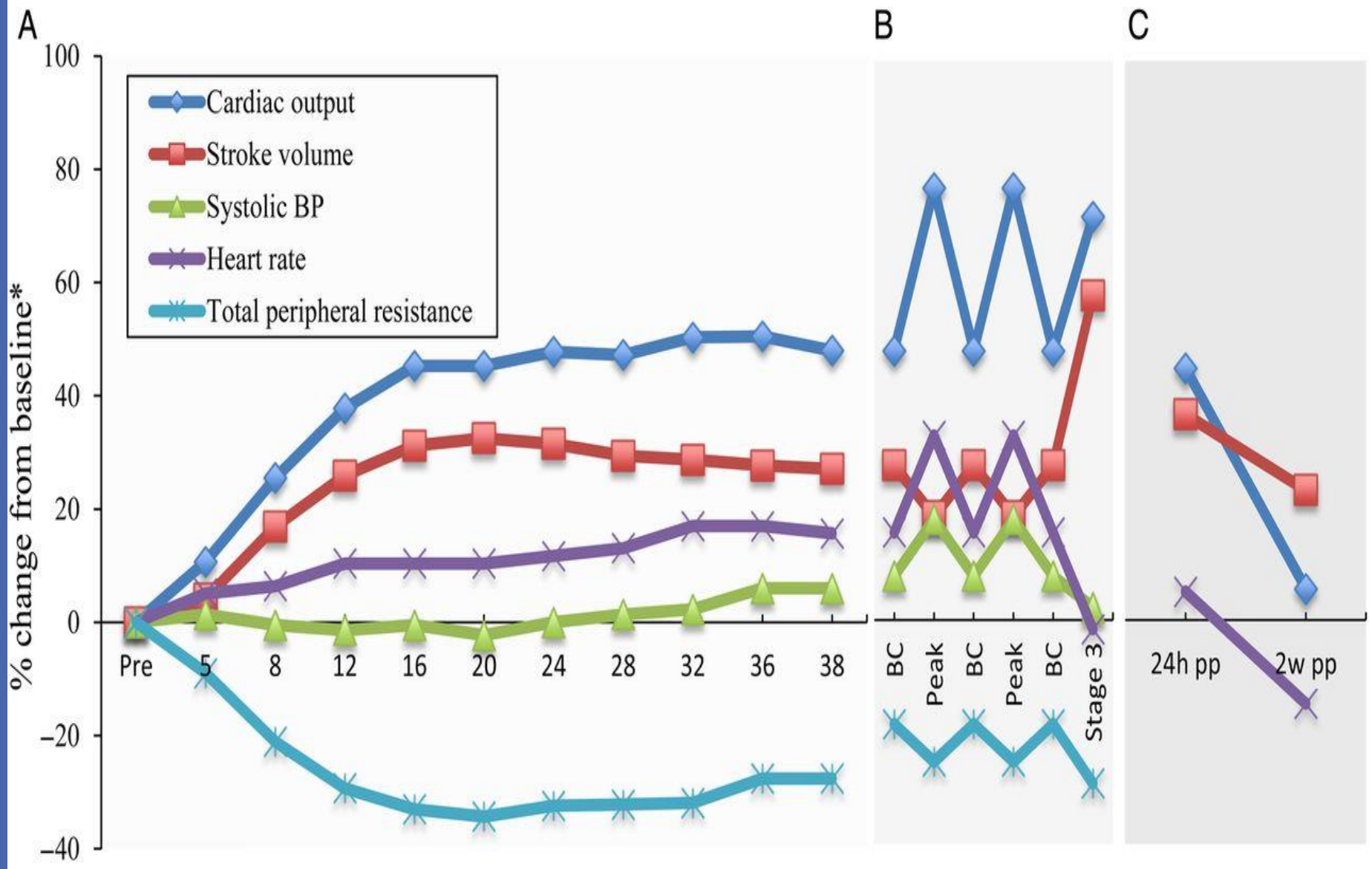


Heart rate

- ↑ 10 – 20 %
- remains high 2–5 d after delivery

SV

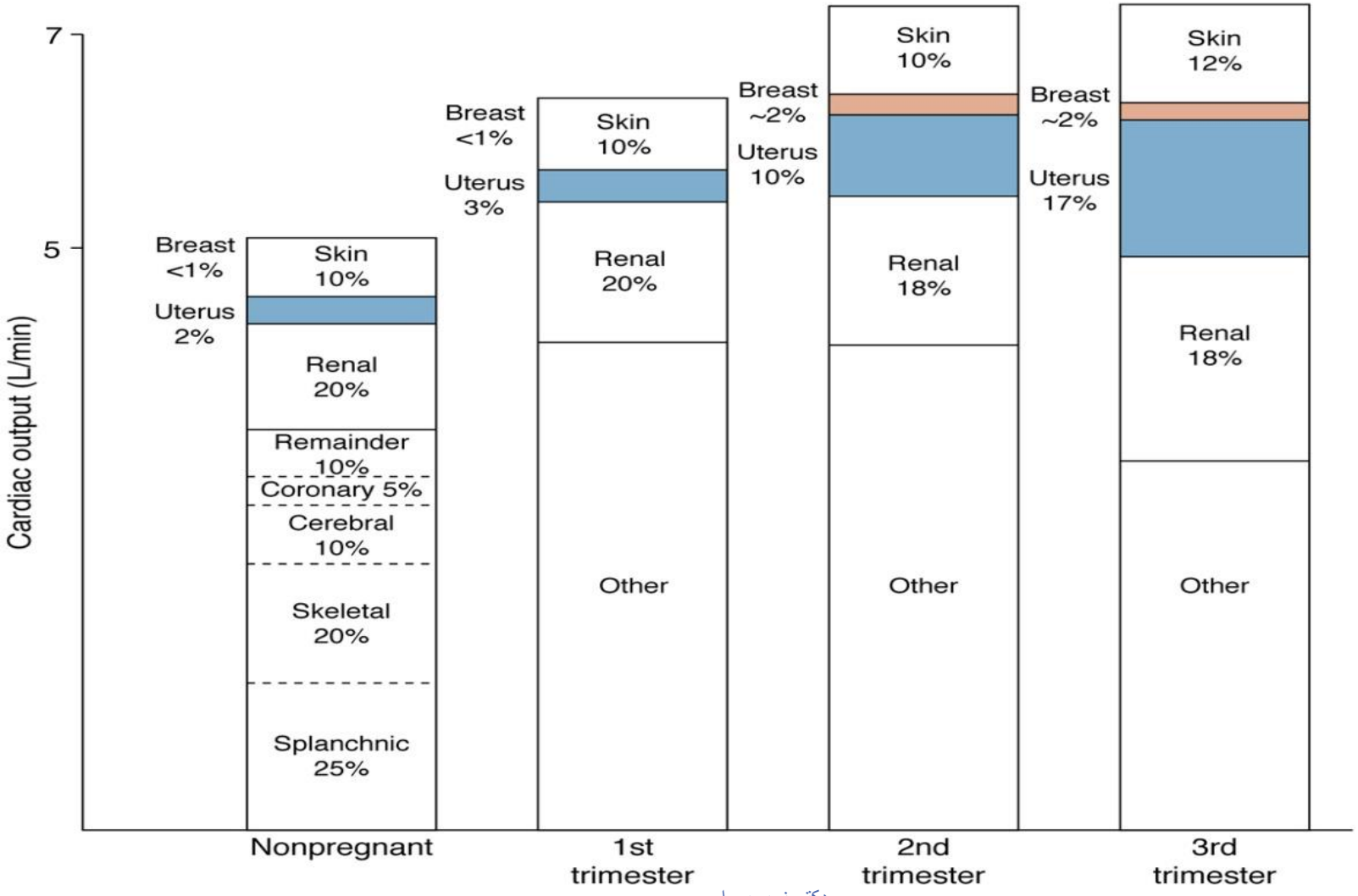
- ↑ from 8 wks
- Peak at 20 wks
- ↓ to baseline by 2 wks PP


A
B
C
100
80
60
40
20
0
–20
–40
% change from baseline*
Cardiac output
Stroke volume
Systolic BP
Heart rate
Total peripheral resistance
Pre
5
8
12
16
20
24
28
32
36
38
BC
Peak
BC
Peak
BC
Stage 3
24h pp
2w pp

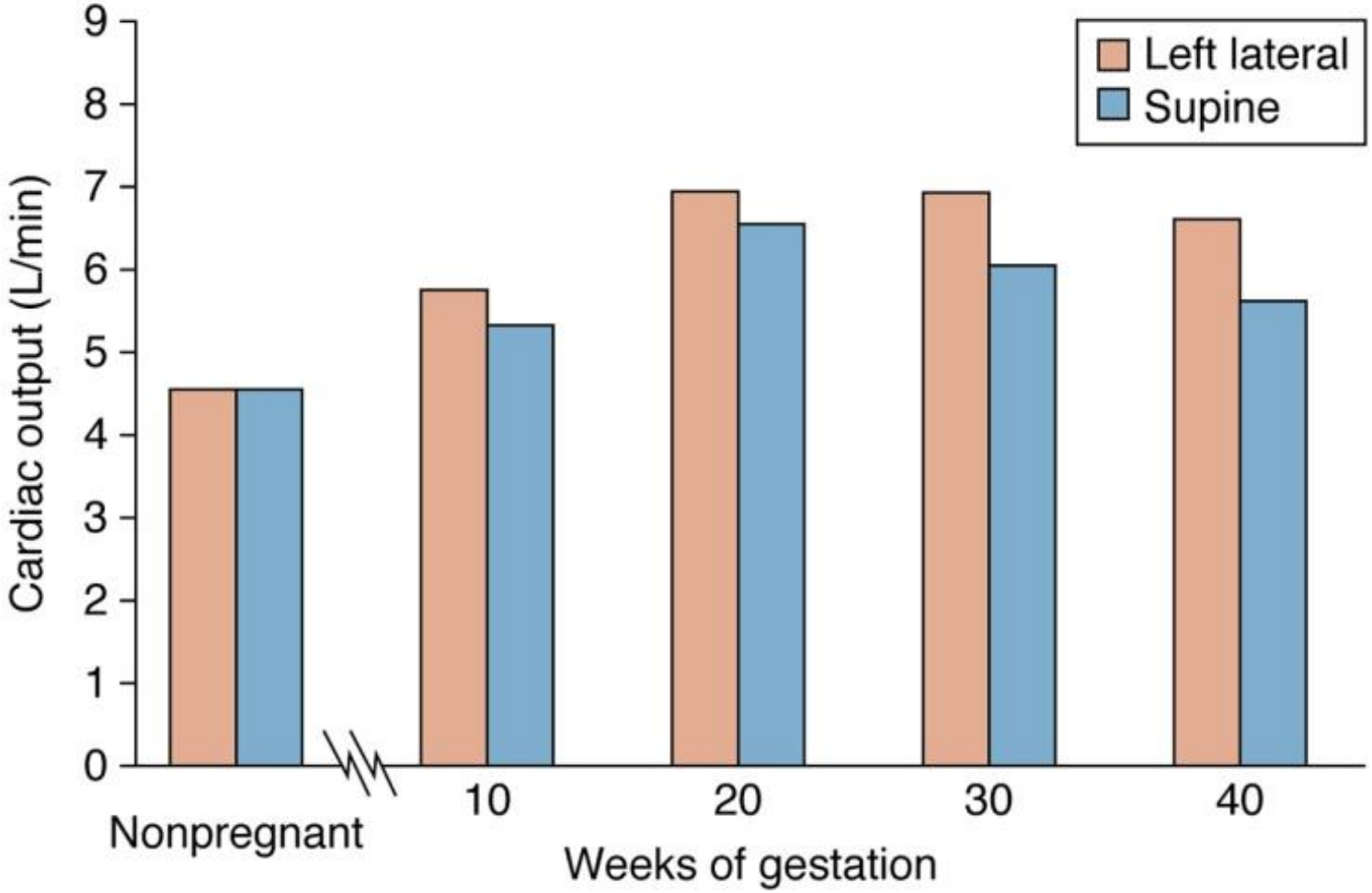
CARDIAC OUTPUT

- CO begins to ↑ in 1st trimester
- By end of 2nd trimester 30-50% above baseline.
- In early pregnancy ↑ CO is primarily by ↑ in SV
- In late pregnancy : HR is the major factor

CO Distribution



CO Variation with position



CARDIAC OUTPUT

- Beginning of labor : > 7 L/min
- Uterine contraction : > 9 L/Min
- Anesthesia : < 8 L/min

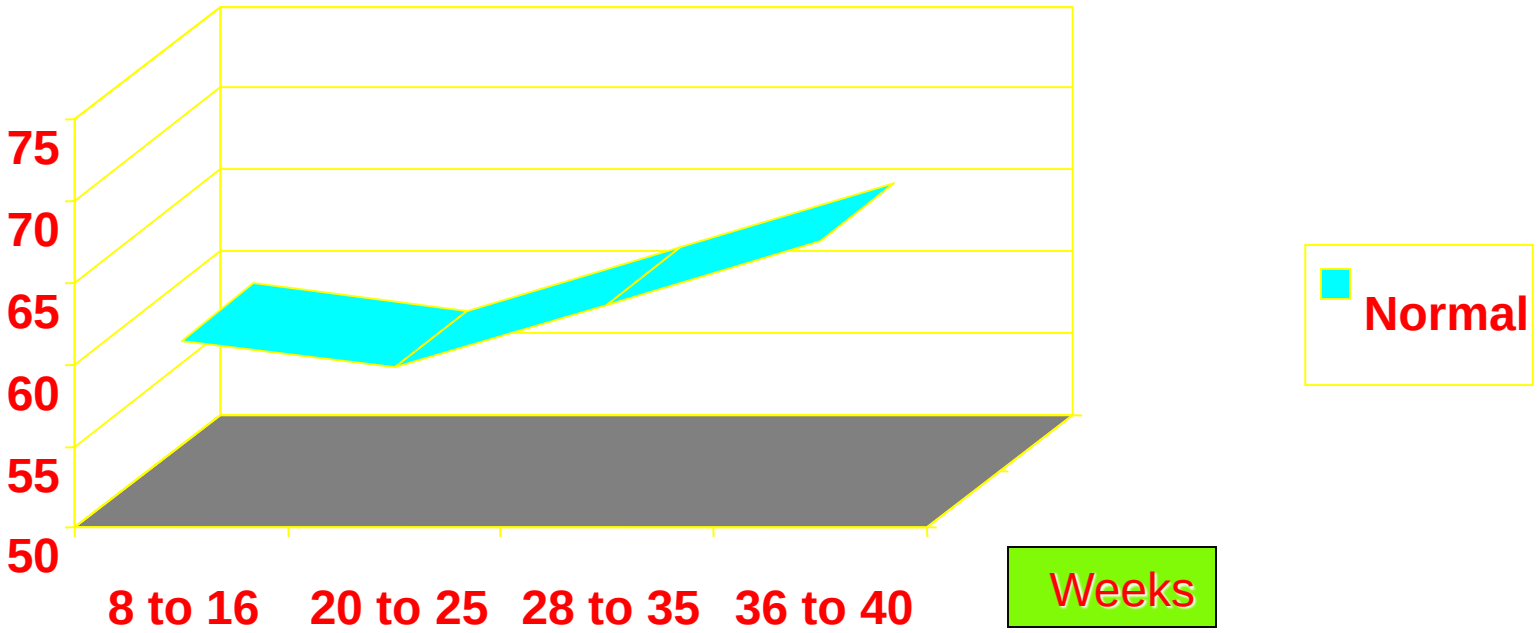
- CO falls to non pregnant values in few wks after delivery

- CO ↑ in twins or triplets is only slightly greater than in single pregnancy

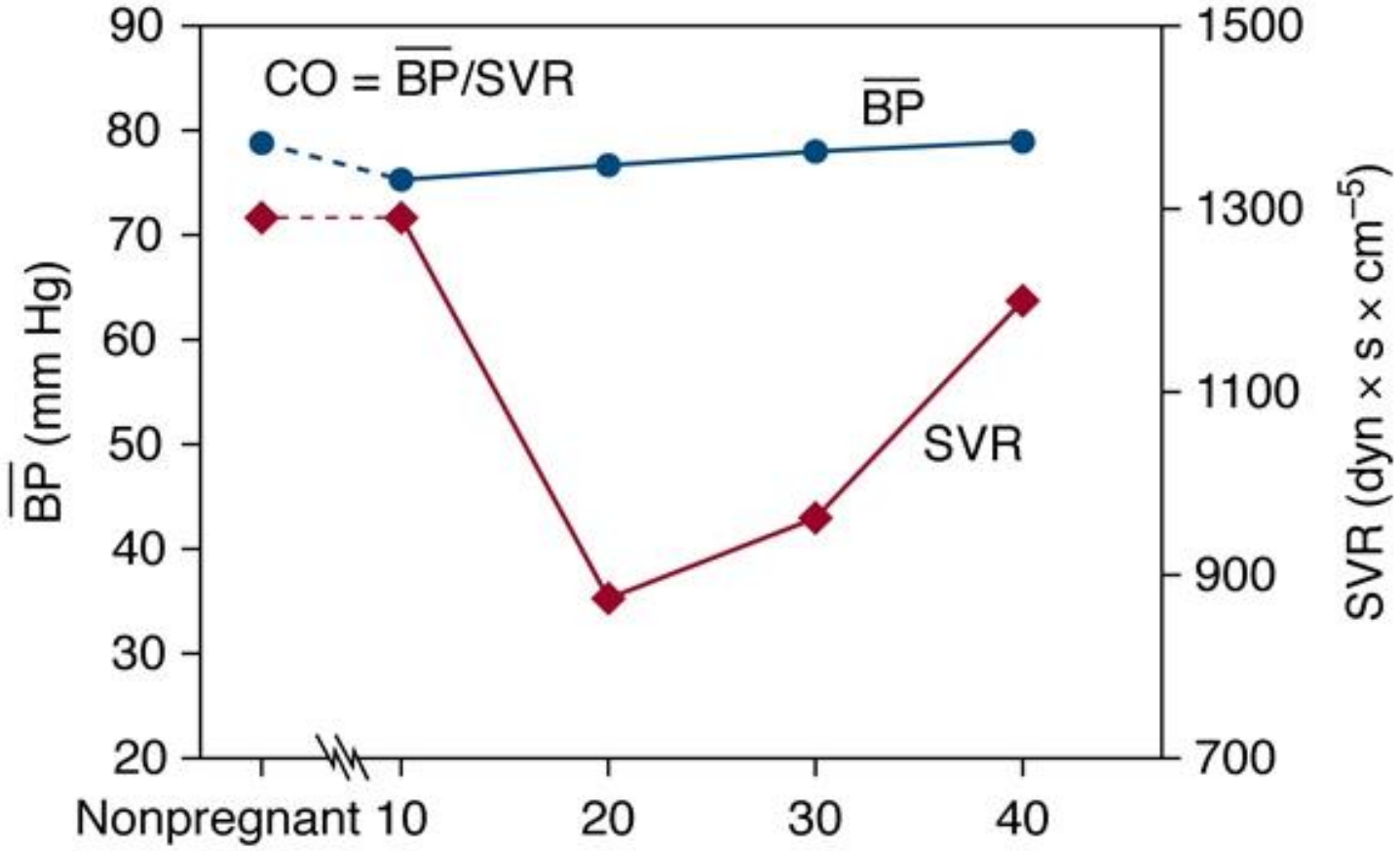
BP

- **BP falls in early gestation & DBP ↓10 mm below baseline in the 2nd trimester**
- **Vasodilatation by prostacyclin & NO**
- **In 3rd trimester DBP ↑ to non-pregnant values by term**

Blood Pressure



BP & SVR


CO = $\overline{BP}$/SVR
$\overline{BP}$
SVR
$\overline{BP}$ (mm Hg)
SVR (dyn × s × cm^{-5})
90
80
70
60
50
40
30
20
1500
1300
1100
900
700
Nonpregnant
10
20
30
40

Pregnancy Adaptations

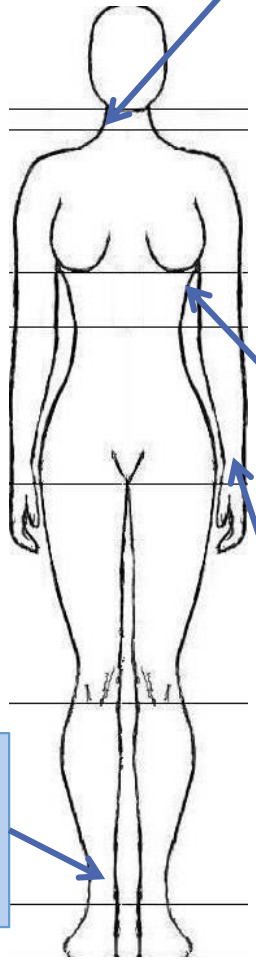
Factor	Preg.	NonPrg	Change
CO	6.2	4.3	+43%
MAP	86	90	-10%
SVR	1210	1530	-21%
PVR	78	119	-34%
HR	83	71	+17%

Mechanisms for Hemodynamic Changes

- Total body water ↑ by 6 to 8 L
- Sodium retention
- Increased arterial compliance
- Increased venous capacitance

Clinical Findings in Normal Pregnancy

Elevated JVP [↑plasma vol]



Apex slightly left & up , prominent impulse

Tachycardia
low DBP
PP ↑ [bounding pulses]

Pedal oedema :
↑ plasma vol &
venous pressures

Changes

- Cardiac axis displaced cephalad and left
  - PMI lateral & elevated (not just due to baby!)
    - Altered thoracic dimensions
  - Left axis deviation
- Murmurs > 96%
  - Virtually all valves
    - Esp. Aortic and Pulmonary
- Rate – increased
- Ventricular distention – 25% increase

ECG

- **Tachycardia**
- **LAD : elev. Diaphragm**
- **Increased ventricular voltage**

Hematologic

- Hypercoagulable
  - Estrogen & Vascular stasis
  - Increased risk for thromboembolic disease
    - Increase in fibrinogen, all coag factors except II, V, XII
    - Fall in protein S and sensitivity to APC
- Fall in platelets and factor XI and XIII
- Increase in WBC

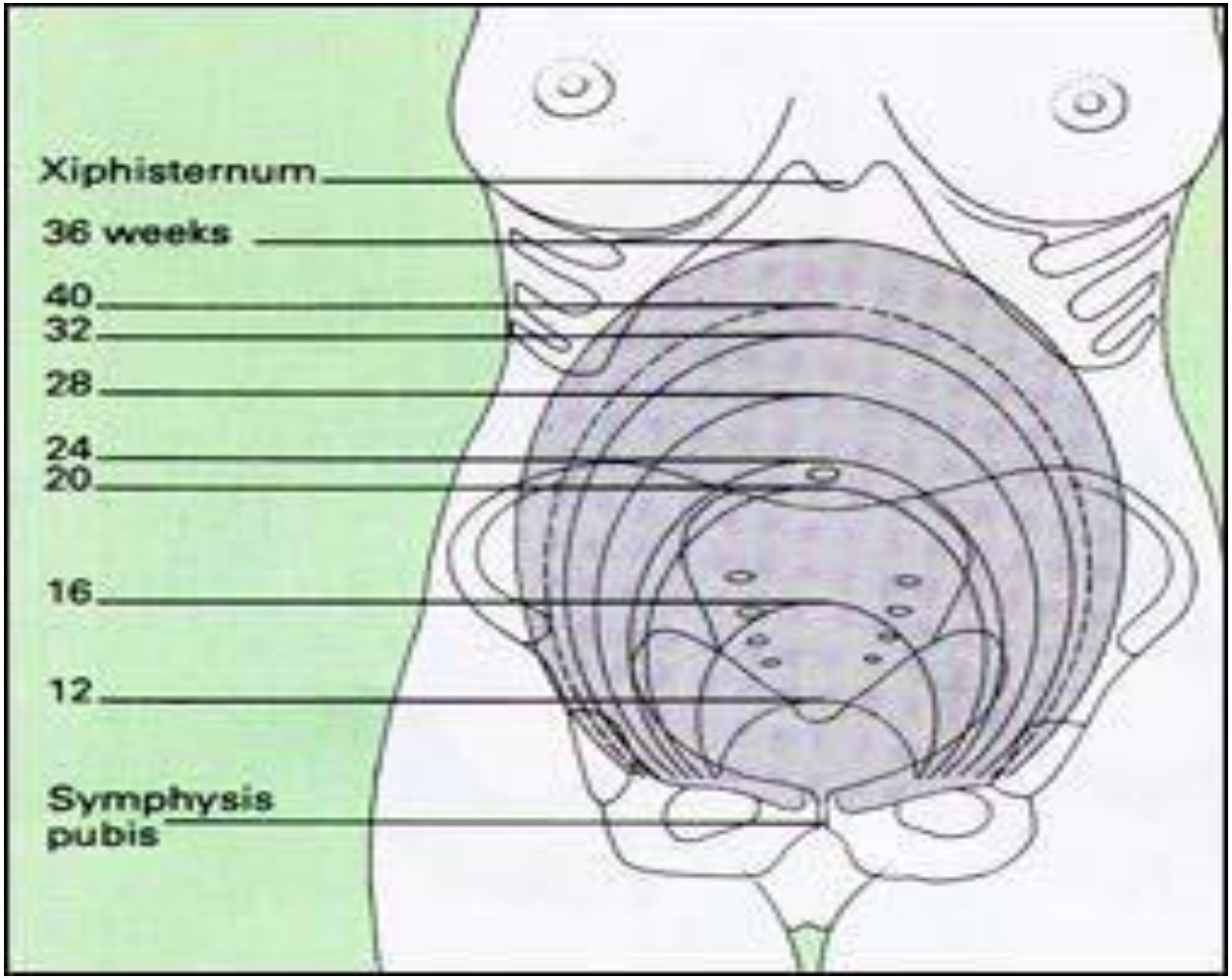
Hematologic changes at term:

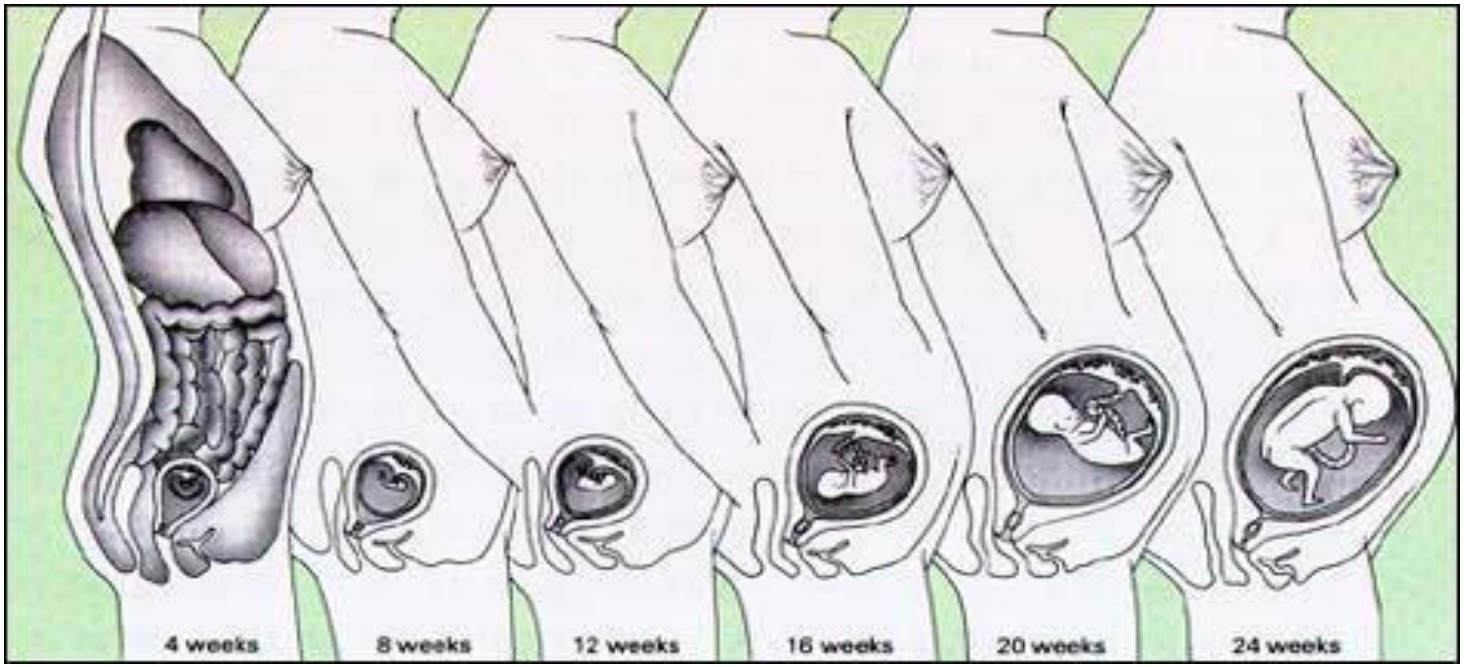
- Fibrinogen increased.
- PT, PTT shortened 20%.
- Increased platelet turnover.

- Increase in coagulation factors,
- immobilization and aorto-caval compression all increase risk of DVT.

ANATOMICAL CONSIDERATIONS

Uterine Position over Time





NORMAL PHYSIOLOGY OR DISEASE?

Signs & Symptoms of Normal Pregnancy that may Mimic Heart Disease

- Signs
  - Peripheral edema
  - JVD
- Symptoms
  - Reduced exercise tolerance
  - Dyspnea
- Auscultation
  - S_3 gallop
  - Systolic ejection murmur
- Chest x-ray
  - Change in heart position & size
- EKG
  - Nonspecific ST-T wave changes
  - Axis deviation

OTHER SYSTEMS

Changes in the Filter

- Renin – stimulated by progesterone
  - Also made by placenta
  - Angiotensinogen → Angiotensin I → Angiotensin II → Aldosterone → Distal tubule
    - Net absorption of Na^+
    - Excretion of K^+
    - Water retention: **6-8 liters**
- Increased renal blood flow
  - 50-75% increase
  - GFR – 50% increase
  - Decreased Albumin = lower colloid oncotic pressure

Other urinary tract changes

- Ureteral dilation / hydroureter
  - Smooth muscle relaxation
  - Later exacerbation by uterine obstruction
  - Urinary stasis*
- Dilation of pelves and calyces
- Increased kidney size

LUNGS AND RESPIRATION

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Respiratory Adaptations

- No change in rate or IRV
- Thorax
  - Tr. Diameter 2cm; circumference 5-7cm
- Increased minute ventilation
- Reduced FRC – 20%
- Increased Tidal Volume – 30-40%
- Compensated respiratory alkalosis
  - pH 7.4+
  - ↑PaO_2; ↓$PaCO_2$ (40 – 30)

At term, mother has respiratory alkalosis with metabolic compensation

ABGs	Non-pregnant	At term
PaCO2	40 ⟶	**30**
PaO2	100	103
pH	7.40	7.44
HCO3-	24 ⟶	**18**

Respiratory Changes

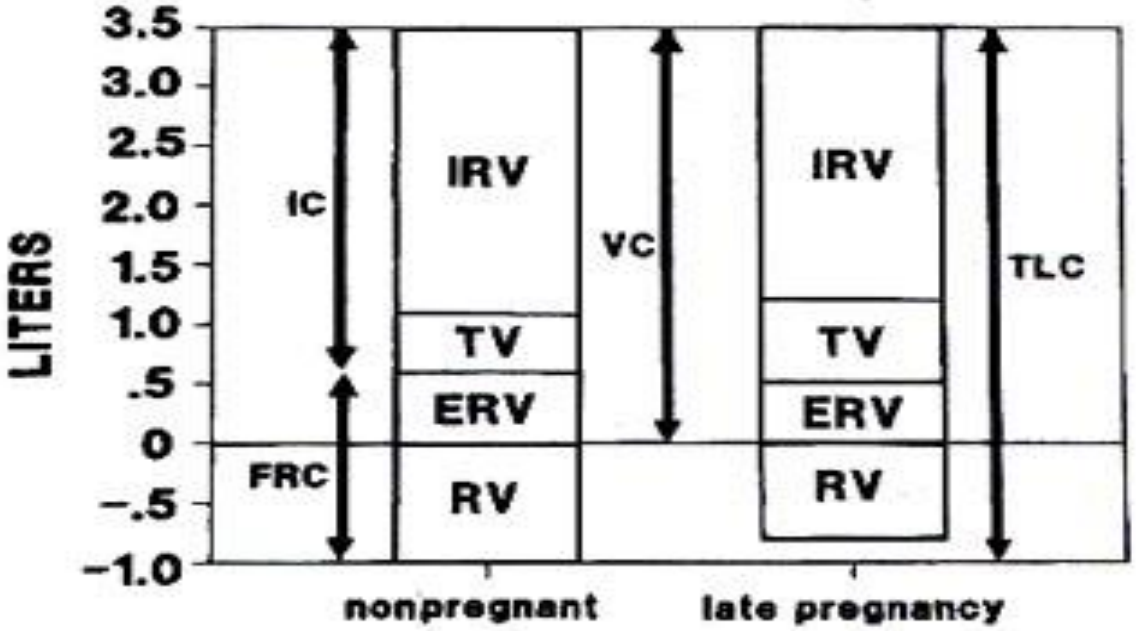

Figure 3–10. Lung volumes in nonpregnant and pregnant women. TLC, total lung capacity; VC, vital capacity; IC, inspiratory capacity; FRC, functional residual capacity; IRV, inspiratory reserve volume; TV, tidal volume; ERV, expiratory reserve volume; RV, residual volume. (From Cruickshank DP, Wigton TR, Hays PM: Maternal physiology in pregnancy. *In* Gabbe SG, Niebyl JR, Simpson JL [eds] Obstetrics: Normal and Problem Pregnancies, 3rd ed. New York, Churchill Livingstone, 1996, p 94, with permission.)

Gastrointestinal

- Slowed GI motility
  - Constipation, early satiety
- Relaxation of LES
  - GERD
- Nausea / vomiting
  - Often proportional to HCG level
- Liver / gallbladder
  - Biliary stasis, cholesterol saturation
    - More stones
  - Coagulation factors
  - Increased binding proteins (thyroid, steroid, vitamin D)

Other "Adaptations"

- "I can't see my feet!!!"
  - Altered center of gravity
  - Altered gait
  - Greater joint laxity
    - Widening of symphysis pubis
    - Affects other joints
    - Thorax; widened costovertebral angle
  - Fatigue / somnolence

Other Changes

- Spider angiomata and palmar erythema
- Hair growth (abdomen and face)
- Mucosal hyperemia
- Striae gravidarum
- Hyperpigmentation (esp. linea nigra)
  - Rashes and acne relatively common

Other Endocrine

- Pancreas
  - Carbohydrate metabolism -Insulin resistance
    - Human placental lactogen, cortisol
- Thyroid Function
  - Increased TIBG (via liver)
  - Increased total T_4 and T_3
    - free levels unchanged
    - HCG suppresses TSH
- Adrenal function
  - Free plasma cortisol is elevated
    - CRH from placenta stimulates ACTH

Pregnancy – not a disease

- Profound changes in physiology and anatomy
- Affects most organ systems
- Can dramatically impact disease states, susceptibility, and treatment
- Almost all will encounter and treat pregnant women
  - Even if you don't know it
- Under-appreciation of changes will lead to suboptimal treatment or outright mistakes

Table 1-2. Differential Diagnosis and Complications of Acute Fatty Liver of Pregnancy

Disease State	Initial Signs and Symptoms	Later Signs and Symptoms	Laboratory Abnormalities	Maternal Complications andComments
Acute fatty liver of pregnancy	Malaise, epigastric pain, nausea/ vomiting, jaundice	Encephalopathy, renal failure, DIC, hypoglycemia	Elevated white blood cell count; elevated ALT, AST, and bilirubin; normal LDH; low platelet count	Pancreatitis, diabetes insipidus, fulminant hepatic failure, sepsis; may also have HELLP syndrome
Severe preeclampsia	Headache, right upper quadrant pain, edema, hypertension	Hypertensive emergency, pulmonary edema	Mildly elevated ALT, proteinuria	Eclampsia, acute renal failure, hepatic capsular rupture
HELLP syndrome	Right upper quadrant pain, nausea/ vomiting, malaise, headaches, visual changes	Hematuria, petechiae, ecchymosis	Low platelet count, elevated bilirubin, elevated AST, ALT, LDH	DIC, acute renal failure, hepatic hemorrhage or failure, ARDS, sepsis, cerebrovascular accident
Viral hepatitis	Nausea, vomiting, fever, jaundice	Encephalopathy, sepsis, coagulopathy, hypoglycemia	Very elevated transaminases, elevated bilirubin, positive serology	Fulminant hepatic failure, hepatic coma, sepsis, GI bleeding
TTP	Mild fever, nausea/ vomiting, abdominal pain, petechiae	CNS effects – headache, visual changes, confusion, seizures; other organ involvement	Very low platelet count and hematocrit, very elevated LDH and bilirubin, mildly elevated ALT, AST	Diffuse subcortical microvascular disease, acute renal failure; could cause placental thrombosis
Antiphospholipid antibody syndrome	Thrombosis	Thrombotic renal disease, GI ischemia	Low hematocrit and platelet count, presence of LA, aCL, or β_2-GP-I	Multiorgan failure leading to "catastrophic antiphospholipid syndrome," development of TTP

aCL = anticardiolipin antibodies; ALT = alanine aminotransferase; ARDS = adult respiratory distress syndrome; AST = aspartate aminotransferase; β_2-GP-I = β_2-glycoprotein-I antibodies; CNS = central nervous system; DIC = disseminated intravascular coagulation; GI = gastrointestinal; HELLP = hemolysis,elevated liver enzymes, and low platelet count; LA = lupus anticoagulant; LDH = lactate dehydrogenase; TTP = thrombotic thrombocytopenic purpura.

Information from: Ko HH,Yoshida E. Acute fatty liver of pregnancy. Can J Gastroenterol2006;20:25–30; and SibaiBM. Imitators of severe preeclampsia. Obstet Gynecol2007;109:956–66.